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Inheritance of the Bark Reaction Resistance Mechanism in *Pinus monticola* Infected by *Cronartium ribicola*

Ray J. Hoff[1]

ABSTRACT

*Necrotic reactions in branch or main stems of western white pine (*Pinus monticola *Dougl.) caused by infection by the blister rust fungus (*Cronartium ribicola *J.C. Fisch. ex Rabenh.) are a major mechanism of resistance. Overall, 26 percent of the seedlings eliminated the fungus via this defense system. Heritability based upon crossing family groups averaged 33 percent for three sets of crossing groups. Heritability based upon individuals averaged 4 percent. The largest genetic advance (selected population compared to the original population before introduction of blister rust) could be made by selecting the average family out of the plus tree selection group. Moderate gains can be made just by selecting the best family. A small but significant gain can be made by selecting seedlings within families. Several breeding methods are discussed concerning the use of bark reaction resistance in new cultivars of western white pine.*

KEYWORDS: *Pinus monticola, Cronartium ribicola,* blister rust resistance

Resistance to blister rust (caused by *Cronartium ribicola* J.C. Fisch. ex Rabenh.) in western white pine (*Pinus monticola* Dougl.) is complex. The complexity is largely due to the varied types of tissues that the fungus grows through from the needles to the stem.

The fungus can enter the stem directly (Van Arsdel 1968), but the usual mode of infection is through stomata of secondary needles (Clinton and McCormick 1919; Patton and Johnson 1967). Symptoms of infection first appear as small yellow spots centered on or near a stomate of a secondary needle within 30 days following inoculation. The fungus produces a large mass of mycelium, called a pseudosclerotium, within the needle directly under the needle spot. It then grows down vascular tissue into the short shoot and thence into the stem. If there is no resistance to stop or impede fungus growth within the needles or stem, the seedling will die within a year or so. Resistance appears to occur very soon after spores of the fungus germinate on the surface of the pine needle and at each change of host tissue, for example leaf mesophyll to leaf vascular system, or leaf vascular system to stem vascular system, etc. Several mechanisms of resistance have been observed (Bingham and others 1971; Hoff and McDonald 1980); undoubtedly there are also a great many genes and/or alleles involved.

Bark reactions were one of the first forms of resistance observed in the white pine-blister rust system (Riker and others 1943). Struckmeyer and Riker (1951) did extensive anatomical studies of these reactions in eastern white pine (*Pinus strobus* L.) and concluded that they were due to the production of a wound-periderm. The more susceptible a seedling, the more extensive mycelium growth was prior to the development of the wound-periderm. No wound-periderm was observed in susceptible seedlings.

Bark reactions have also been reported for sugar pine (*Pinus lambertiana* Dougl.) (Kinloch and Littlefield 1977) and Armand pine (*Pinus armandii* Franch.) (Hoff and McDonald 1972). Boyer (1964) also reported bark reactions for eastern white pine. In a study of 16 white pine species, Hoff and others (1980) reported bark reactions in all species except eastern white pine.

[1]Principal plant geneticist located at Intermountain Station's Forestry Sciences Laboratory, Moscow, ID.

This publication reports on the heritability of a resistance reaction in the stem—that is, bark reactions—that eliminates the fungus at various times after it has entered the stem.

Because much of the variation in bark reaction data was due to differences between full-sib families, single gene inheritance patterns were sought. No such patterns were found, however, so this character was treated as polygenic, and genetic gains are presented accordingly. This approach is probably more logical because bark reactions do vary in size and time of action in western white pine (fig. 1). The small reaction in figure 1A occurs shortly after the fungus enters the stem, and the reaction in figure 1D occurs 2 to 3 or more years after stem entry.

MATERIALS AND METHODS

Blister-rust-free parent trees were selected for this test. The selections came from throughout the range of western white pine in northern Idaho and western Montana. In all cases, the selected trees were surrounded by blister-rust-killed or heavily infected trees. Frequently, the mortality within the stand was over 90 percent. Lowest was about 40 percent.

The mating design was similar to the factorial design of experiment II of Comstock and Robinson (1952). Bingham and others (1969) discuss assumptions pertaining to this design to breeding for resistance to blister rust in western white pine. The progeny were planted in a randomized complete-block design. Each cross was represented by a 16-seedling plot (two seedlings by eight) in each of six blocks.

In addition, 10 control seed collections were taken from the major rust resistance selection areas. The control cones were either from squirrel caches or from several rust-infected trees. However, these selection areas have had from 40 to 90 percent mortality as a result of blister rust and, therefore, the controls (survivors) probably contain more resistance genes than did the original population prior to blister rust introduction.

Data in this paper were based on the performance of progenies from three groups of testers crossed with selected trees. Each crossing group had four different testers and there were 51 selections in group I, 21 in group II, and 17 in group III. Testers were usually male parents and selected female parents, but in several cases, when there were not enough female strobili on the selected tree, the reciprocal cross was made. But because previous unpublished work indicated no reciprocal or maternal effects, the testers and selections were analyzed without regard to their role as male or female parent.

Test seedlings were inoculated in the fall, usually in September, after their second growing season, that is, a 2-year-old seedling, under conditions outlined for previous tests by Bingham (1972). The inoculum was obtained from heavily infected leaves of *Ribes hudsonianum* var. *petiolare* (Dougl.) Jancz. growing along the west fork of Hobo Creek about 16 km northeast of Clarkia, ID.

Rust inspection was completed as described by Hoff and McDonald (1980). The following data were collected:
(1) 9 months after inoculation—presence of needle spots;
(2) 12 months after inoculation—presence of needle spots and stem symptoms (normal cankers plus bark reactions);
(3) 24, 36, and 48 months after inoculation—presence of stem symptoms.

Analysis of the data was completed after the fourth-year rust inspection and was based on the number of seedlings that were healthy because of a bark reaction that eliminated the fungus divided by the total number of seedlings with stem symptoms, including those that had died due to blister rust, 4 years after inoculation. Those seedlings with some kind of needle resistance were therefore not included.

The statistical model assumes that the selected trees used as males and females were random samples from the resistant tree populations. This is probably valid for all trees except group I males. These trees had been selected for higher than average rust resistance from a previous test. They were, however, selected for a high level of total resistance and not specifically for bark resistance.

The method of analysis followed Bingham and others (1969), Becker and Marsden (1972), and Becker (1971). The formula for the model was:

$$X_{ijk} = \mu + M_i + F_j + (MF)_{ij} + R_k + b_{ijk} + e_{ijk} + d_{ijk}$$

where

X_{ijk} = the transformed proportion of healthy seedlings with bark reactions from the cross of the ith male and the jth female in the kth replication

μ = general mean

M_i = the effect of the ith male, $i = 1, 2, \ldots I$

F_j = the effect of the jth female, $j = 1, 2, \ldots J$

$(MF)_{ij}$ = the effect of the interactions of the ith male and the jth female

R_k = the effect of the kth replication, $k = 1, 2, \ldots k$

b_{ijk} = effect of binomial sampling

e_{ijk} = effect of plot

d_{ijk} = effect of individuals within plots.

Plot means were transformed (arcsin $\sqrt{\text{percent}}$) and missing values were estimated using the method of Steel and Torrie (1960, page 130).

The analysis of variance, expected mean squares, and formulas for estimating the variance components and standard errors are shown in table 1.

Heritability was calculated in two ways—one based on individual seedlings, the second on the selection unit, which is the full-sib family unit (Bingham and others 1969). The formulas were as follows, on an individual seedling basis:

$$h^2_{\text{Ind}} = \frac{\sigma^2_A}{\sigma^2_F + \sigma^2_M + \sigma^2_{MF} + \sigma^2_e + \frac{1}{w}\sigma^2_b - \frac{1}{w}\sigma^2_d}$$

where σ^2_A is the additive genetic variance, estimated from $4\,\sigma^2_F$ or σ^2_M. On a family unit basis:

$$h^2_{\text{fam}} = \frac{\sigma^2_A}{\sigma^2_F + \frac{\sigma^2_M}{I} + \frac{\sigma^2_{MF}}{IJ} + \frac{\sigma^2_e + \frac{1}{w}\sigma^2_b - \frac{1}{w}\sigma^2_d}{K}}$$

Table 1—Model for analysis of variance, expected mean squares, variance components, and standard errors

Source of variation	d.f.*	Mean squares	Expectation of mean squares
Replications	$K-1$	MS_R	
Males	$I-1$	MS_M	$\sigma_e^2 + \frac{1}{w}\sigma_b^2 - \frac{1}{w}\sigma_d^2 + K\sigma_{MF}^2 + KJ\sigma_M^2$
Females	$J-1$	MS_F	$\sigma_e^2 + \frac{1}{w}\sigma_b^2 - \frac{1}{w}\sigma_d^2 + K\sigma_{MF}^2 + KI\sigma_F^2$
Male × female	$(I-1)(J-1)$	MS_{MF}	$\sigma_e^2 + \frac{1}{w}\sigma_b^2 - \frac{1}{w}\sigma_d^2 + K\sigma_{MF}^2$
Male-female × replications	$(IJ-1)(K-1)$	MS_{MFR}	$\sigma_e^2 + \frac{1}{w}\sigma_b^2 - \frac{1}{w}\sigma_d^2$

*I, J, and K = total numbers of testers, candidates, and replications, respectively. Individual variance components are:

$\sigma_M^2 = \frac{MS_M - MS_{MF}}{KJ}$ = variance due to males

$\sigma_F^2 = \frac{MS_F - MS_{MF}}{KI}$ = variance due to females

$\sigma_{MF} = \frac{MS_{MF} - MS_{MFR}}{K}$ = variance due to interaction of males and females

$\sigma_e^2 - \frac{1}{w}\sigma_d^2 = MS_{MFR} - \frac{1}{w}\sigma_b^2$† σ_e^2 = variance due to effect of plot

σ_d^2 = variance due to effect of individuals

$\sigma_b^2 = \frac{1}{w}821$ = variance due to effect of binomial sampling

w = harmonic mean number of seedlings

S.E. $= \sqrt{\frac{2}{C^2}\sum\frac{MS_n^2}{fn+2}}$ = standard errors

†Becker and Marsden 1972.

††C = coefficient of the variance component, MS_n = the nth mean square used to estimate the variance component, and fn = the degrees of freedom of the nth mean square.

where σ_A^2 is equal to 2 σ_F^2, being one-half of the additive genetic variance.

Successive genetic gains were calculated beginning with the amount of resistance in the base populations—that is, all western white pines in the area sampled prior to the introduction of blister rust.

The formula for genetic gain is $\Delta G = Sh^2$, where S is the selection differential expressed as the mean of the individuals selected for the next generation minus the overall mean. The h^2 on female family was used with this formula to determine the gain from selection.

When the best individuals within the best families were selected, the selection differential was not known. The genetic gain was estimated by the formula $\Delta G = Sh^2\sigma$ phen, where S is the standardized selection differential (Becker 1967, table II) and σ phen is the phenotypic standard deviation. The h^2 based on individuals was used for this gain. The resulting gain was added to the mean calculated from transformed observations, and then converted back to percent. The percent gain was this total minus the percent mean of the population.

RESULTS

Average percentage of healthy seedlings with bark reactions ranged from 20.2 to 29.4 for male groups. The range for all females was 8.0 to 44.2 percent. Controls averaged 14.2 percent (table 2).

Analysis of variance of the plot values, transformed (arcsin $\sqrt{\text{percent}}$), is shown in table 3. Mean squares for females were significant in all three crossing groups. Mean squares for males were significant for group III. Variance components are shown in table 4 with standard errors.

Table 5 displays heritabilities based on a selection unit basis (full-sib family) and on an individual basis. The male × female interaction estimates one-quarter of the dominance and one-eighth of the epistatic variance, so the values in table 4, group I and II, cannot be negative in a biological sense. In determining heritabilities, these values were assumed to be zero.

Genetic gains from three main sources could be made: (1) increase in resistance over the base population (base population here is defined as the population prior to the

Table 2—Results of artificial inoculation of seedlings in three western white pine crossing groups

Component			Crossing groups I	II	III
Average seedling number/plot			8.43	9.24	8.04
Total seedlings			10,318	4,657	3,280
Average percent healthy					
	Males	a	32.2	19.7	45.3
		b	29.8	19.1	27.3
		c	29.3	18.6	24.3
		d	26.3	17.6	20.8
Group average, percent healthy			29.4	20.2	29.4
Range of candidates, percent healthy			43.1-14.9	32.7-8.0	44.2-19.2
Controls, percent healthy	Average			14.5	
	Range			32.3-8.5	

Table 3—Analysis of variance of transformed data

Source of variation	Group I df†	Group I ms	Group II df†	Group II ms	Group III df†	Group III ms
Replications	5	8,599.07**	5	3,669.22**	5	2,052.30**
Males	3	385.18	3	616.52	3	4,063.69**
Females	50	539.84**	20	612.31**	16	526.10*
Male × female	150	277.98	60	260.23	48	397.15
Male – female × replications	1,009	326.32	412	264.99	331	332.44

†Degrees of freedom were reduced for missing plots as follows: 6 in group I, 3 in group II, 4 in group III.
*Significant at the 10 percent level of probability.
**Significant at the 1 percent level of probability.

Table 4—Variance components for percent bark reactions in three western white pine crossing groups

Variance factors and harmonic mean	Group I	Group II	Group III
σ_M^2	0.35 ± 0.80*	2.83 ± 1.27*	35.95 ± 25.20*
σ_F^2	10.91 ± 4.61	14.67 ± 3.25	5.37 ± 8.02
σ_{MF}^2	−8.06 ± 5.84	−4.76 ± 3.61	10.78 ± 13.92
$\sigma_e^2 - \frac{1}{w}\sigma_d^2$	183.79	140.22	167.91
$\frac{1}{w}\sigma_b^2$	142.53	124.77	164.58
Harmonic mean, w	5.76	6.58	4.99

*Standard error.

Table 5—Estimates of genetic variance and heritabilities of bark reactions in three western white pine crossing groups

Populations	Crossing group I			Crossing group II			Crossing group III		
	σ_A^2	σ_D^2	h^2	σ_A^2	σ_D^2	h^2	σ_A^2	σ_D^2	h^2
Individual male	1.40	0	0.1 ± 0.3*	11.32	0	1.2 ± 1.3*	143.80	43.12	14.1 ± 9.7*
Individual female	43.64	0	4.3 ± 1.8	58.68	0	6.0 ± 3.3	21.48	43.12	2.1 ± 3.1
Selection unit (full-sib family)	21.82		0.33 ± 0.14	29.34		0.49 ± 0.27	10.75		0.15 ± 0.23

*Standard error.

introduction of blister rust) by using all families; (2) increase in resistance through the selection of best families; and (3) selection of individuals within best families.

The gain in resistance over the base population of an average family in group I is expected to be 29.4 percent. This could be an overestimate because the males were selected for a high level of total resistance that probably included some bark resistance. For group II the gain is estimated as 20.2 percent and for group III, 29.4 percent.

The second increment of gain is the selection of the best families. This gain ranged from 2.3 to 6.2 percent when the five best families were selected. Means for the top 20 percent female families are listed in table 6. The gains are shown in table 7 under ΔG_F for various selection levels.

The third gain comes from selecting individuals within the best family and is listed under ΔG_{Ind} in table 7. This gain ranged from 0.9 to 2.7 percent within the top five families.

Total gain is the sum of the three gains, and they are listed under total gain above base in table 7. For the top five families, the gains were 35.4 percent for group I, 29.1 percent for group II, and 32.6 percent for group III.

Table 6—Bark resistance of the top 20 percent of selected western white pine trees

Group	Female family identification	Overall mean
		Percent
I	32	43.1
	260	42.5
	95	41.3
	115	40.5
	194	40.4
	274	39.3
	31	39.0
	220	38.7
	258	38.6
	34	37.2
II	247	32.7
	133	32.3
	132	28.5
	281	27.6
	173	26.5
III	373	44.2
	326	39.7
	348	38.6
	312	36.1
	100	35.5

Table 7—Expected genetic gain (percent) from crossing superior families or superior seedlings of these families at various selection intensities

Families selected	Group I			Group II			Group III		
	ΔG_F†	ΔG_{Ind}*	Total gain** above base	ΔG_F	ΔG_{Ind}	Total gain above base	ΔG_F	ΔG_{Ind}	Total gain above base
5	4.3	1.7	34.4	6.2	2.7	28.1	2.3	0.9	31.6
10	4.1	1.9	34.4	6.1	2.7	28.1	1.9	.9	31.2
25	3.5	1.9	33.8	4.6	2.9	26.7	1.6	.9	30.9
50	2.1	2.1	32.6	2.7	3.0	24.9	1.1	.9	30.4

†Genetic gain from selective mating of selected families.
*Genetic gain from selection and mating of seedlings from selected families.
**Includes genetic gain of average family over base population (1 percent resistance): 28.4 for group I; 19.2 for group II; and 28.4 for group III.

DISCUSSION

An average of 26 percent of the seedlings, over all three groups, produced necrotic reactions in stem tissues that prevented the fungus from further growth (table 2). On the other hand nearly 15 percent of the control seedlings also developed bark reactions that eliminated the fungus. So what is the gain in resistance? Bingham and others (1960) used the control data to adjust the level of resistance in the selected population. At that time total resistance of the controls was 5.3 percent. Total resistance (needle resistance plus bark resistance) of the controls in our test was 22.8 percent, reflecting an increase in resistance that is associated with the increase in mortality of white pine trees within the natural stands.

The controls cited in the present paper were survivors in stands that have had as much as 93 percent mortality due to blister rust. Therefore, they probably carry several resistance genes—possibly not enough to survive to rotation, but enough so that when crossed with other survivors, including the resistance candidates in the stand, a relatively high level of resistance is expressed in their progeny. The question really centers on the amount of resistance in original population, that is, the population before blister rust was introduced. In an experimental planting of 500 3-year-old western white pine, Mielke (1943) reported that 6 years after planting only nine seedlings were alive, but five were infected; and within another 2 years all were infected. One of the stands that

Figure 1—Bark reactions in western white pine in response to blister rust: (A) Reaction soon after fungus enters stem. (B and C) Reaction after fungus has grown in stem for a while. (D) With this reaction fungus growth and reaction alternate—that is, the fungus grows followed by a bark reaction that does not completely kill the fungus.

produced seed for this bark reaction study was first infected in 1937 when about 15 years old. When the seed was collected for this test—1964—the stand had undergone 93 percent mortality (345/371 trees per acre) due to blister rust. By 1984, mortality was 98 percent and based on the condition of remaining trees will likely increase to over 99 percent in 2 to 3 years. Therefore, for this study, resistance in the base population was fixed at 1 percent. Still, this is a conservative figure to use for bark resistance because the 1 percent includes all mechanisms of resistance.

The differences among the full-sib families leave little doubt that substantial gains can be made after just one selection cycle. This original selection was a combination of natural selection by infecting and killing of the most susceptible trees and of artificial selection by our choosing trees with no infections among the survivors. Stem resistance was increased from 1 percent (resistance in the base population) to about 25 percent—averaged over the three groups—with just this one cycle of selection.

Predicting additional gain for future selection was not nearly as good. Table 7 indicates that an average of just 4 percent could be made by choosing the best five families. Gain by selecting the best individuals resulted in an increase of less than 2 percent. Several reasons for these low additional gains are evident:

1. The low selection differential; there were only 89 candidate trees, and these were separated into three breeding units (selection groups). In surveys of natural stands Bingham (1983) concluded that in the high hazard stands 1 tree in 10,000 was not infected; thus the first round of selection was based on very high selection differential.
2. In each family seedlings were few and varied in number. Seedlings with needle resistance—resistance factors that function before stem resistance (Bingham and others 1971)—were removed from the data set. Because needle resistance is highly heritable some families were left with only a few individuals, some with many.
3. Variation in bark resistance is high. Bark reaction varied from small to very large (fig. 1). The fungus was often killed quickly, whereas at other times the fungus lingered on, disappeared, and sometimes reappeared. Then too, some seedlings showed a well-defined bark reaction along with a typical susceptible reaction. The reactions were often separated; therefore, it was obvious that they came from separate primary infections, namely, needle spots. This is typical of a differential reaction that occurs when there is genetic variation in the fungus. This results in confusion and error in the resistance rating system.
4. The infection load was uneven. Although all seedlings that were included in this study were infected, some had many separate primary infections (100 or more needle spots) whereas other seedlings had only one.

Despite difficulties with this data, the relatively large gain that can be made by just selecting average families would seem to justify using stem resistance in seed orchards. Choosing a portion of the best families may, with luck, even provide a little more gain. The level of stem resistance is not high enough to use by itself for most planting sites, and should not be used singly anyway because of the threat of new blister rust races, but when used in combination with other mechanisms of resistance it will likely provide adequate levels of resistance. At the same time, the combination of several resistance factors would provide defense against new races of blister rust.

For advanced breeding programs much more knowledge is needed. Two important objectives must be addressed: (1) to determine if the observed bark reaction differences reflect different mechanisms and/or genes; (2) to determine the relationship of present observed races of the blister rust fungus with the various bark reaction types.

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324 25th Street
Ogden, UT 84401

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